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Publisher Psychology Press

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## Multivariate Behavioral Research

Publication details, including instructions for authors and subscription information:

<http://www.informaworld.com/smpp/title~content=t775653673>

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Online Publication Date: 01 July 2009

**To cite this Article** King, Daniel W., King, Lynda A., McArdle, John J., Shalev, Arieh Y. and Doron-LaMarca, Susan(2009)'Sequential Temporal Dependencies in Associations Between Symptoms of Depression and Posttraumatic Stress Disorder: An Application of Bivariate Latent Difference Score Structural Equation Modeling',Multivariate Behavioral Research,44:4,437 — 464

**To link to this Article:** DOI: 10.1080/00273170903103308

**URL:** <http://dx.doi.org/10.1080/00273170903103308>

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# Sequential Temporal Dependencies in Associations Between Symptoms of Depression and Posttraumatic Stress Disorder: An Application of Bivariate Latent Difference Score Structural Equation Modeling

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Depression and posttraumatic stress disorder (PTSD) are highly comorbid conditions that may arise following exposure to psychological trauma. This study examined their temporal sequencing and mutual influence using bivariate latent difference score structural equation modeling. Longitudinal data from 182 emergency room patients revealed level of depression symptom severity to be positively associated with changes in PTSD intrusion, avoidance, and hyperarousal over 3 time intervals, beginning shortly after the traumatic event. Higher scores

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on depression anticipated increases (or worsening) in PTSD symptom severity. The pattern of influence from PTSD symptom severity to change in depression symptom severity simply followed the general trend toward health and well-being. Results are discussed in terms of the dynamic interplay and associated mechanisms of posttrauma depression and PTSD symptom severity.

Perhaps the most studied mental health consequences of psychological trauma are depression and posttraumatic stress disorder (PTSD), two conditions that are often highly comorbid in trauma victims. As a part of the National Comorbidity Survey, Kessler, Sonnega, Bromet, Hughes, and Nelson (1995) reported approximately 48% of those with a lifetime diagnosis of PTSD also experienced at least one lifetime major depressive episode. Men with PTSD were almost seven times more likely to have a major depressive episode than men without PTSD; the comparable odds ratio for women was just above four. In an urban community sample of young adults, Breslau, Davis, Andreski, and Peterson (1991) found 37% of those with current PTSD met criteria for major depression. Similarly, Breslau, Davis, Peterson, and Schultz (1997) estimated a 43% lifetime comorbidity rate for major depression and PTSD among a large sample of new mothers drawn from two hospitals in one metropolitan area. Data from the National Vietnam Veterans Readjustment Study (Kulka et al., 1990) suggested that 44% of male Vietnam veterans with a lifetime diagnosis of PTSD also have a lifetime diagnosis of major depression, and 56% of female Vietnam veterans with a lifetime diagnosis of PTSD also have a lifetime diagnosis of major depression. Among male and female Vietnam veterans with current PTSD, 16% and 23%, respectively, had current depression. High rates of co-occurrence have been reported for a variety of other specific trauma populations (e.g., Blanchard, Buckley, Hickling, & Taylor, 1998, for motor vehicle accident casualties; Norris, Murphy, Baker, & Perilla, 2004, for flood victims; North, Smith, & Spitznagel, 1997, for survivors of a mass shooting).

Beyond the establishment of the co-occurrence of depression and PTSD—and the assertion of reasonable common or higher order factors that might explain their comorbidity (e.g., Brown, Chorpita, & Barlow, 1998; Koenen et al., 2003; Krueger, 1999; Watson, 2005)—there is concern for the temporal sequencing of these two disorders and/or their symptoms. Following exposure to a traumatic event, do depression and PTSD ensue simultaneously? Does depression symptomatology precede PTSD symptomatology? Are PTSD symptoms antecedent to depression symptoms? Is there a complex interplay between the two conditions, such that high or low symptom severity for one portends a subsequent spike or decrement in symptom severity for the other? An understanding of the dynamics of the depression-PTSD association can inform mechanisms, and thus etiology and, ultimately, effective treatment development. If one can uncover patterns of mutual influence on shifts in symptom severity that underlie or structure

observed covariation or individual differences in comorbidity, one can better anticipate exacerbations of symptoms for either condition and tailor interventions accordingly.

Prior research addressing the temporal sequencing of depression and PTSD varies with regard to the basic design used: cross-sectional with current and retrospective reports of mental health status (e.g., Breslau et al., 1997; Kessler et al., 1995; Skodol et al., 1996) versus the more informative longitudinal approach with prospective data over two or more occasions (e.g., Breslau, Davis, Peterson, & Schultz, 2000; D. J. Erickson, Wolfe, King, King, & Sharkansky, 2001; McFarlane & Papay, 1992; Shalev et al., 1998). They also differ with regard to their conclusions about temporal sequencing. For example, based on patterns of findings from the cross-sectional National Comorbidity Survey, Kessler et al. concluded that PTSD was probably antecedent to its comorbid conditions, including depression. Breslau et al. (2000) used both the National Comorbidity Survey data and data from their own prospective community sample of young adults to assert the temporal precedence of PTSD. On the other hand, longitudinal data analysis from Shalev et al.'s (1998) community sample of emergency room admittees did not support a PTSD-to-depression pathway, although reports of depression prior to the traumatic event were predictive of the later occurrence of both depression and PTSD. Breslau et al. (1997) obtained lifetime psychiatric histories from their large sample of women and found that a primary diagnosis of PTSD was associated with increased risk for first-onset major depression, and pretrauma major depression was a risk factor for both trauma exposure and subsequent PTSD, given trauma exposure. These latter findings rely on retrospective accounts of onset and also are somewhat equivocal concerning temporal precedence.

Finally, D. J. Erickson et al. (2001) used a prospective cross-lagged panel design to examine associations between symptoms of depression and PTSD in Gulf War I veterans. Controlling for gender, combat exposure, and survey administration (in-person, mail, or telephone), they endorsed a model with bidirectional and positive cross-lagged influences for total scores on depression and PTSD. When parallel analyses were conducted for depression with each of three features of PTSD (intrusion or reexperiencing the traumatic event via dreams, thoughts, and feelings; avoidance of event reminders and emotional numbing; and hyperarousal), similar bidirectional associations were found for intrusion and avoidance-numbing, but only early hyperarousal predicted later depression with null findings for the reverse direction.

In any panel design, in which a variable is measured over multiple occasions, there are two possible internal mechanisms or sources of change that must be taken into account before the influence of another variable can be evaluated. The first and most obvious is the influence of scores on that same variable on a prior occasion, termed the *autoregressive* effect (see Gollob & Reichardt, 1987).

D. J. Erickson et al.'s (2001) use of a cross-lagged panel design to examine the depression-PTSD linkage accommodates such autoregressive effects, with the partialled cross-lagged coefficients representing the association between the predictor (PTSD or depression at Time 1) and the outcome (depression or PTSD at Time 2). Here, change in the outcome of interest is expressed as the difference between the observed outcome score and that which would be predicted from status on that variable on the previous occasion. Thus, the partialled cross-lagged coefficient is the association between the antecedent and this residual "change."

Yet, the logic for this prediction of residual change rests on the assumption that there is no second internal source of potential change, change due to a natural course (possibly a function of unknown or unspecified factors). In the time series literature, *stationarity* is demonstrated by the equality of means and variances of the outcome over multiple assessments (Browne & Nesselroade, 2004). Although stationarity is not uncommon, in some instances it is not reasonable to anticipate such an assumption to hold. One example is the natural increase, and then late-life decrease, in cognitive ability as a function of age (McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002). In the current context, a natural tendency to wellness for trauma victims is expected and has been demonstrated (Gilboa-Schechtman & Foa, 2001; King, King, Salgado, & Shalev, 2003; Koss & Figueredo, 2004). Therefore, *nonstationarity* as a contributor to an expected general trend toward recovery also must be appraised and, if present, controlled, to avoid confounding in the interpretation of partialled cross-lagged coefficients. In other words, cross-lagged panel models account for changes in rank order but not changes in means over time. Where mean changes are important (as they often are in mapping depression or PTSD symptom severity postexposure), cross-lagged models may not be the most appropriate.

With reference to the outcome variable, an alternative to the use of residual change scores is the use of direct change scores themselves. Simple difference or gain scores have been espoused by Nesselroade and Cable (1974), Rogosa, Brandt, and Zimowski, (1982), and Williams and Zimmerman (1996). Others (e.g., Cronbach & Furby, 1970; Lord & Novick, 1968) have argued against direct change scores, largely on the basis of their putative unreliability and associated invalidity. Such arguments about unreliability and invalidity can be circumvented by adopting latent variable modeling strategies and incorporating measurement error, thereby rendering optimally reliable differences between perfectly reliable latent variables.

The goal of this study was to build upon prior work and attempt to clarify the temporal sequencing of symptoms of depression and PTSD using an innovative longitudinal prospective methodology that is uniquely suited to representing dynamic change. By dynamic change, we refer to a psychological process in which one's status on an individual differences characteristic is altered over time

due to one or more purported “forces” or potentially explanatory intrapersonal or contextual variables. Trauma research is especially amenable to dynamic change models due to the time-based link of posttrauma consequences to the traumatic event itself. There is a beginning, at the moment of the event, from which symptoms can be charted and described as a function of time, and individual differences in the relationship between time and symptom severity become worthy of investigation. Regarding the co-occurrence of depression and PTSD, both are subject to the process of posttrauma dynamic change, each potentially influencing change in the other over time. When both are measured on multiple occasions, depression may be considered a time-varying or time-dependent covariate in the prediction of change in PTSD, and PTSD may be considered a time-varying or time-dependent covariate in the prediction of change in depression.

Thus, we proposed that dynamic change in symptom severity following trauma exposure can best be demonstrated by change scores (and not residual change), in particular, by change scores derived from perfectly reliable latent variables. In addition, the two internal mechanisms of change, autoregressive effects and nonstationarity or natural growth/decline, must be specified and statistically controlled, where appropriate, before the role of any antecedent on change is evaluated. The methodology used in this study, dynamic change analysis via latent difference scores (Hamagami & McArdle, 2001; L. A. King, King, McArdle, et al., 2006; McArdle, 2001; McArdle & Hamagami, 2001), is designed to take these concerns into consideration. Our global research question was *What is the dynamic interplay between depression and PTSD symptom severity following exposure to a traumatic event?* In addressing this question, we acknowledged the rather extensive literature (e.g., Amdur & Liberzon, 2001; Asmundson et al., 2000; DuHamel et al., 2004; D. W. King, Leskin, King, & Weathers, 1998; Palmieri, Weathers, Difede, & King, 2007; Simms, Watson, & Doebbeling, 2002) that supports a multifactorial first-order structure of PTSD rather than a single first-order PTSD factor or a higher order PTSD construct. Accordingly, we addressed the research question by examining bidirectional associations between symptoms of depression and each of four PTSD symptom factors: (a) intrusion (reexperiencing the event in one’s feelings, thoughts, and dreams), (b) avoidance (efforts to circumvent reminders of the event), (c) numbing (emotionally detaching from one’s relations and environment), and (d) hyperarousal (extreme vigilance, irritability, and exaggerated startle). This classification scheme largely reflects the official B, C, and D symptom categories defined by the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV)* (American Psychiatric Association, 1994) yet also accommodates the disaggregation of the C category into separate effortful avoidance and emotional numbing clusters, a conceptualization that is empirically well supported (L. A. King, King, Orazem, & Palmieri, 2006).

## METHOD

### Data Source and Sample

Data were taken from a prospective study conducted by Shalev and colleagues (Shalev et al., 2008) at a large medical center in Jerusalem. The Shalev data are comprised of information on emergency room patients who experienced a PTSD Criterion A traumatic event (American Psychiatric Association, 1994) and then were followed longitudinally. Traumatic events included motor vehicle accidents, work and domestic accidents, physical assaults, terrorist acts, and war-related events. Participants initially were assessed at the hospital shortly following the index event, then again at three subsequent assessments targeted at 1 week, 1 month, and 4 months following exposure. Posttraumatic mental health outcomes were assessed at each follow-up occasion using self-report and clinical interview measures. The sample of size  $N = 182$  was comprised of 106 (58%) men and 76 (42%) women. Age ranged from 16 to 65 years ( $M = 31.16$ ,  $SD = 11.30$ ). Participants were predominantly single (48%) or married (43%), and their education consisted primarily of partial high school (19%), completed high school (38%), or bachelor's degree (22%).

### Measures

**Beck Depression Inventory (BDI).** At each assessment occasion, participants' depression symptoms were measured using a Hebrew version of the BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a 21-item self-report instrument. For each item, there are four possible responses indicating the present intensity of depression symptoms. Responses are scored on a 0- to 3-point scale ranging from nondepressed (e.g., *I am not particularly discouraged about the future*) to severely depressed (e.g., *I feel that the future is hopeless and that things cannot improve*). Acceptable internal consistency reliability has been reported for the BDI ( $\alpha = .86$ ; Beck & Steer, 1984), and the scale is a valid, widely recognized, and extensively used measure of depression in studies of psychopathology (Beck, Steer, & Garbin, 1988; Richter, Werner, Heerlein, Kraus, & Sauer, 1998). The Hebrew version was the product of the expected systematic procedure of translation and back-translation, has proved reliable and valid in an extensive program of research (Shalev et al., 1998), and generated a coefficient alpha of .91 in the present study.

**Impact of Event Scale-Revised (IES-R).** The IES-R (Weiss & Marmar, 1997) is a 22-item self-report questionnaire derived from statements most frequently used by people to describe recently experienced distressing events. The IES-R was revised from the original 15-item Impact of Event Scale (IES;

Horowitz, Wilner, & Alvarez, 1979) to better address *DSM-IV* PTSD criteria. Creamer, Bell, and Failla (2003) found the internal consistency reliability of full scale scores to be .96; Weiss and Marmar (1997) reported strong internal consistency reliability for the PTSD symptom clusters ranging from the mid .80s to low .90s. Evidence for the validity of the IES-R may be found in its associations with a variety of other self-report and interview-based measures of PTSD (e.g., Corapcioglu, Yargic, Geyran, & Kocabasglu, 2006; S. J. Erickson & Steiner, 2000; Paunovic & Ost, 2005). In the present study, respondents were asked to indicate on a 4-point scale (0 = *not at all*, 1 = *rarely*, 3 = *sometimes*, and 5 = *often*) the frequency of each symptom during the previous 7 days. The Hebrew version of this instrument was informed by a systematic translation and back-translation from the original English version, and it has been employed successfully in contemporaneous applications with Israeli trauma victims (Shalev & Freedman, 2005). Based on Amdur and Liberzon's (2001) confirmatory factor analysis of the IES and accumulating evidence that the avoidance and numbing features of PTSD are best represented as separate entities (L. A. King, King, Orazem, et al., 2006), the IES-R items were classified into four symptom clusters representing the intrusion, avoidance, numbing, and hyperarousal features of PTSD. For the present study, coefficient alphas were as follows: IES-R total: 22 items, .94; IES-R intrusion: 8 items, .90; IES-R avoidance: 5 items, .78; IES-R numbing: 3 items, .46; IES-R hyperarousal: 6 items, .83.

*Clinician-Administered PTSD Scale (CAPS).* Posttraumatic stress symptoms also were measured with a Hebrew version of the CAPS (Blake et al., 1990). The CAPS is a structured interview for use by mental health professionals to assess the 17 PTSD symptoms as established in the *DSM-IV*. The frequency and intensity of each symptom are measured using a 5-point (0 to 4) Likert-type rating scale, and severity scores (0 to 8) may be computed by summing the frequency and intensity ratings for each symptom. A total PTSD severity score may be obtained by summing across all 17 symptoms; symptom cluster severity scores likewise may be computed. Blake et al. (1995) reported internal consistency reliability of .94 for the total 17-item scale, and .85 to .87 for the three *DSM-IV* symptom clusters (i.e., intrusion, avoidance-numbing, and hyperarousal). Additionally, Blake et al. (1995) reported sound convergent validity for the CAPS with other recognized measures of PTSD. The Hebrew version of the CAPS, used here, was subjected to standard procedures to ensure cross-cultural equivalence and has shown strong evidence for reliability and validity (Shalev et al., 1998). As with the IES-R, the present study disaggregated the avoidance-numbing symptom category into separate clusters. Coefficient alphas were as follows: CAPS total: 17 items, .90; CAPS intrusion: 5 items, .73; CAPS avoidance: 2 items, .52; CAPS numbing: 5 items, .77; CAPS hyperarousal: 5 items, .75.

**Latent difference score model.** The longitudinal paradigm guiding analyses in this study was McArdle's latent difference score structural equation model for dynamic change (Hamagami & McArdle, 2001; L. A. King, King, McArdle et al., 2006; McArdle, 2001; McArdle & Hamagami, 2001), which offers three benefits: control for autoregressive effects, control for nonstationarity, and optimally reliable change scores to document process. Figure 1 displays a simple latent difference score model for a series of four assessments on a single observed variable, for example, depression. The figure employs a path graphic representation (McArdle & Boker, 1990; McArdle & Hamagami, 2001) wherein *boxes* represent observed variables or manifest indicators, *circles* represent unobserved or latent variables (factors and residuals), *single-headed arrows* reflect regression weights, *double-headed arrows* depict variances or covariances, and the *triangle* is a placeholder to accommodate the estimation of means. Unlabeled single-headed arrows assume a regression weight of 1; labeled arrows are parameters potentially to be estimated. The foundation is a latent or unobserved difference score ( $\Delta d$ ; we use the letters  $d$  and  $D$  to designate interest in change in

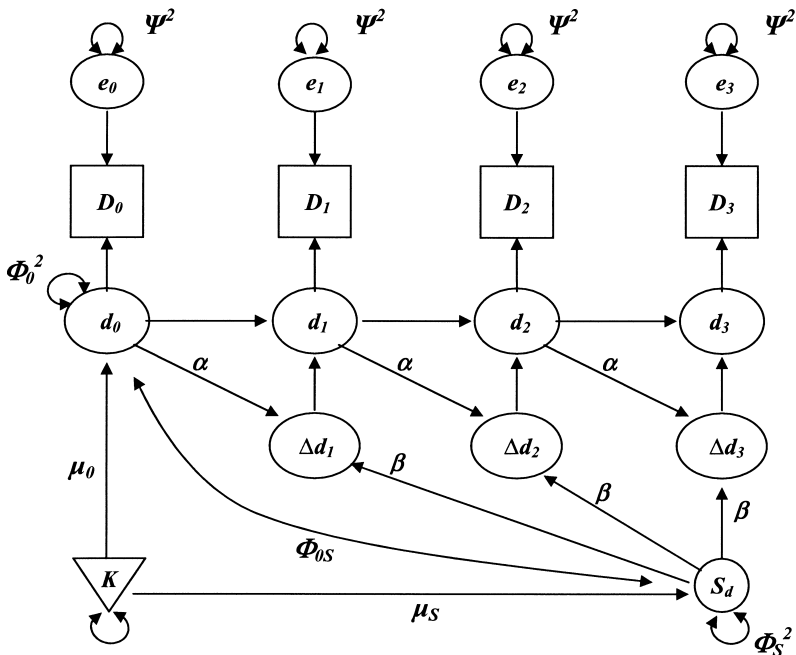


FIGURE 1 Univariate dual change latent difference score model.

depression) adopted to circumvent problems inherent in observed difference scores. The observed variable is  $D$ , assessed on four occasions. Consistent with classical test theory,  $D$  is a composite of two latent variables, true score variable  $d$  and error  $e$ . Hence, we see that the model has observed variables  $D_0$  through  $D_3$ , latent variables  $d_0$  through  $d_3$ , and errors  $e_0$  through  $e_3$ , corresponding to the four assessments of depression.

Extracting equations from the model using standard path analysis conventions, we find that

$$d_1 = d_0 + \Delta d_1,$$

and therefore

$$\Delta d_1 = d_1 - d_0,$$

and thus for  $\Delta d_2$  and  $\Delta d_3$ . As differences between perfectly reliable adjacent true scores, latent difference scores  $\Delta d_1$ ,  $\Delta d_2$ , and  $\Delta d_3$  are optimally reliable (see Williams & Zimmerman, 1996, for equations describing the reliability of difference scores).

The autoregressive effects of prior status on the latent difference scores are depicted by the alphas ( $\alpha$ s), also called proportional change coefficients. The alphas may or may not be constrained to be equal over time. A latent variable or constant change variable ( $S_d$ ) represents nonstationarity or natural change over time. Its effect is symbolized by the betas ( $\beta$ s), which are typically fixed at 1. The model allows for the estimation of means and variances of initial status and constant change ( $\mu_0$  and  $\Phi_0^2$ ,  $\mu_S$  and  $\Phi_S^2$ , respectively) and a covariance between the two ( $\Phi_{0S}$ ). Residual variances of measurement error ( $\Psi^2$ s) are typically constrained to be equivalent.

Thus, in the univariate case, with  $D$  representing observed scores on depression,  $\Delta d_t = \alpha_d d_{t-1}$  [autoregressive effects] +  $\beta_d S_d$  [nonstationarity].

Correspondingly designating observed PTSD symptom severity over time as  $P$ ,  $\Delta p_t = \alpha_p p_{t-1}$  [autoregressive effects] +  $\beta_p S_p$  [nonstationarity].

In the bivariate case involving both depression and PTSD, the model can be expanded to incorporate a system of relationships depicting mutual dynamic change for the two variables:

$$\Delta d_t = \alpha_d d_{t-1} + \beta_d S_d + \gamma_{dp} p_{t-1} \text{ and } \Delta p_t = \alpha_p p_{t-1} + \beta_p S_p + \gamma_{pd} d_{t-1}.$$

Therefore, the bivariate latent difference score approach prescribes change in a variable (depression or PTSD) at any given timepoint as a function of prior status on the other variable (PTSD or depression), controlling for the two internal sources of change for each. The gamma coefficients ( $\gamma_{dp}$  and  $\gamma_{pd}$ )

represent the cross-variable lagged influences of PTSD on change in depression and depression on change in PTSD, respectively. Figure 2 depicts this more complex bivariate latent difference score model, which formed the foundation for the analyses in this study. The core issue is to determine the extent to which one variable of interest influences change in the other variable of interest, and vice versa.

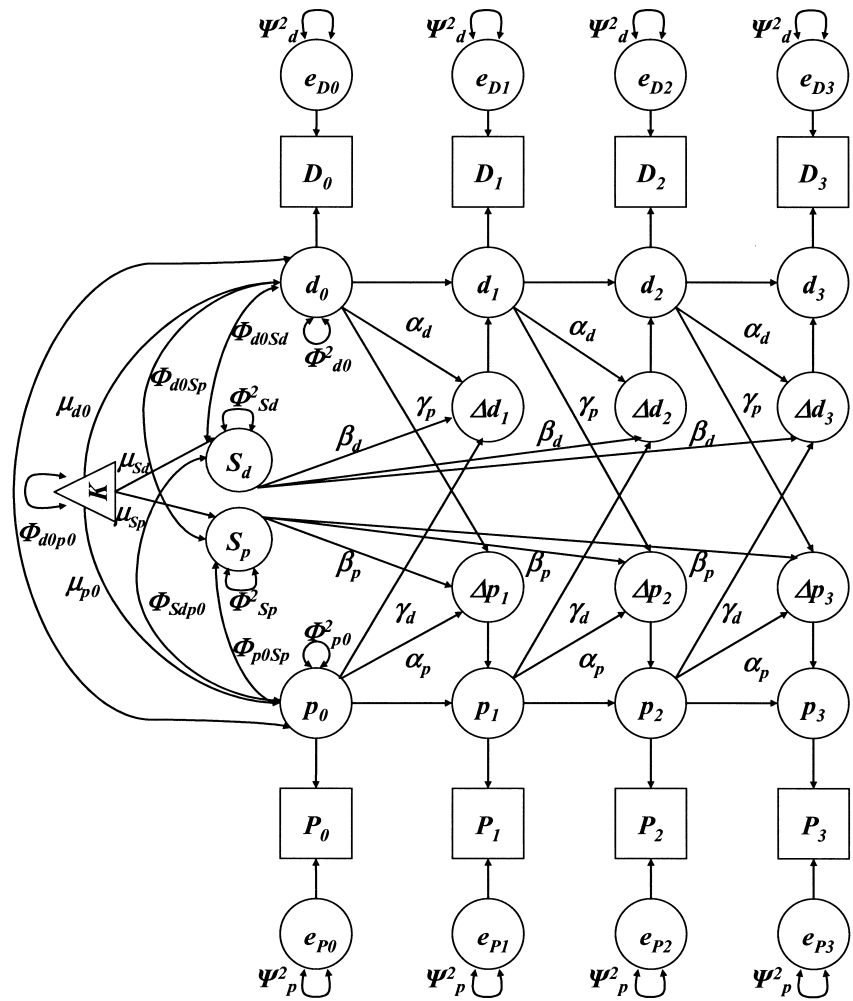


FIGURE 2 Bivariate dual change latent difference score model. To simplify presentation, single-indicator measurement components are depicted.

*Overview of data analyses.* As noted previously, posttrauma depression and PTSD data were targeted for collection at 1 week, 1 month, and 4 months following the emergency room admission. Of the total number of possible assessments ( $182 \text{ participants} \times 3 \text{ occasions} = 546$ ), 450 (82%) were completed. Of the 182 participants, 120 (66%) provided data on all three occasions; 148 (81%) provided data on at least two occasions; and 34 (19%) provided data on only one occasion. With 152 persons assessed at the targeted first occasion, 132 assessed at the targeted second occasion, and 161 assessed at the targeted third occasion, attrition over time was not a particularly salient issue. As with most longitudinal studies of trauma victims (D. W. King et al., 2006), however, there was substantial dispersion in the actual timing of assessments around the targeted timepoints. Therefore, we implemented procedures outlined by D. W. King et al. (2006) wherein depression and PTSD scores for each participant were assigned to four time classes related to the precise count of days since the trauma on which their specific assessments were made. The objective was to minimize the dispersion of participants' time since exposure within time classes and to maximize the number of participants for which covariances were calculated between variables defined by time classes (e.g., depression for Time Class 1 with depression for Time Class 2). Using this approach, each of the 182 participants had scores for depression (BDI), and the four PTSD symptom clusters (intrusion, avoidance, numbing, and hyperarousal) for the two PTSD measures (IES-R and CAPS) systematically placed into one, two, or three of these time classes, depending on the number of assessment occasions on which he or she provided data.<sup>1</sup> Following this data restructuring, we checked for the effects of missingness on outcomes by employing Hedeker and Gibbons's (1997) random-effects pattern-mixture model wherein a missing data dichotomy (all three targeted assessments versus one or two assessments) was regressed on components of the latent growth curves depicting the association between time and scores on the variables of interest.

Using the Mplus software package (Version 5.1; Muthen & Muthen, 1998–2006) and the full information maximum likelihood estimator, means and standard deviations were derived for depression and each of the two sets of PTSD symptom cluster scores (IES-R and CAPS) over the time classes. A series of bivariate latent difference score models (see Figure 2) was evaluated, coupling depression with each of the four PTSD symptom clusters to determine sequential temporal dependencies. Depression had a single indicator, total score on the BDI. For each of the four PTSD symptom clusters, there were two indicators,

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<sup>1</sup>Whereas BDI and IES-R evaluations were a part of all assessment occasions, the CAPS was relegated to only the last two assessment occasions because it requires symptom duration of 1 month and the first assessment was targeted at 1 week following trauma. This situation was readily accommodated by incorporating a missing variable placeholder (McArdle & Woodcock, 1997).

one from the IES-R and the other from the CAPS. The metric for interpreting latent variables for depression was in terms of BDI total scores; the metric for latent variables for PTSD was in terms of each cluster's CAPS summative score. Regarding the measurement component for PTSD, loadings and intercepts were constrained to be equal over occasions, and measurement error variance likewise was constrained to be equal over occasions, both requirements for these longitudinal panel analyses (McArdle, 2007; Meredith & Horn, 2001). In addition, proportional change coefficients consistently were constrained to be equivalent within depression and PTSD constructs, a standard for latent difference score analyses. Cross-variable lagged coefficients were likewise constrained to be equivalent for the models involving depression and PTSD intrusion, depression and PTSD avoidance, and depression and PTSD hyperarousal, but not for the model involving depression and PTSD numbing (further explanation to follow).<sup>2</sup>

## RESULTS

### Descriptive Statistics

The upper rows of Table 1 present information on central tendency and variability for the four time classes that structured the data for the 182 study participants. The four time classes yielded three time intervals upon which the latent difference scores were based: from Time Class 1 to Time Class 2 was the first time interval, with the "middle score" or median days since the event spanning from roughly 9 to 36; from Time Class 2 to Time Class 3 was the second time interval, with median days since the event from approximately 37 to 131; and from Time Class 3 to Time Class 4 was the third time interval, with median days since the event from about 132 to 192. Note that not all 182 cases had an initial assessment within the first 20 days. In addition to some missing data resulting from attrition, the restructuring resulted in incomplete data by design. There were no significant findings in the random-effects pattern-mixture analyses (Hedeker & Gibbons, 1997) that evaluated the possibility of an interaction between missingness and time of assessment in the prediction of scores. Therefore, individual trajectories of symptom severity over the course of the study did not differ for those who provided complete versus those who provided incomplete data. Data were assumed to be missing at random (Little & Rubin, 2002) and amenable to the Mplus full information maximum likelihood estimation procedure.

The lower portion of Table 1 provides maximum likelihood estimates of means and standard deviations for scores on the BDI and the IES-R and CAPS

<sup>2</sup>Mplus scripts for all bivariate latent difference score analyses in this article are available upon request from Daniel W. King.

TABLE 1  
Descriptive Statistics for Time Structure, Depression, and PTSD

	<i>Time Class 1</i> (0–20 Days)	<i>Time Class 2</i> (21–80 Days)	<i>Time Class 3</i> (81–160 Days)	<i>Time Class 4</i> (> 160 Days)
Number of observations	147	117	97	60
Mean days event to assessment	10.16	40.09	132.07	203.97
Median days event to assessment	9.00	36.00	131.00	191.50
Standard deviation	3.40	12.44	14.19	43.80
Range 5th–95th percentile	6–17	27–66	106–155	163–308
BDI total				
<i>M (SD)</i>	11.16 (9.17)	8.04 (8.35)	7.46 (9.49)	7.40 (8.30)
IES-R intrusion				
<i>M (SD)</i>	17.11 (10.75)	12.48 (10.22)	9.68 (10.37)	8.33 (7.61)
IES-R avoidance				
<i>M (SD)</i>	7.84 (6.42)	7.75 (7.10)	6.83 (7.03)	5.57 (5.17)
IES-R numbing				
<i>M (SD)</i>	3.90 (3.37)	3.79 (3.47)	3.08 (3.21)	2.84 (2.73)
IES-R hyperarousal				
<i>M (SD)</i>	11.61 (8.45)	9.84 (7.74)	7.99 (8.08)	7.71 (7.69)
CAPS intrusion				
<i>M (SD)</i>		8.18 (7.50)	5.76 (8.74)	5.39 (6.99)
CAPS avoidance				
<i>M (SD)</i>		3.31 (4.18)	2.67 (4.07)	1.97 (3.28)
CAPS numbing				
<i>M (SD)</i>		5.79 (7.51)	5.78 (8.50)	5.47 (7.94)
CAPS hyperarousal				
<i>M (SD)</i>		9.68 (8.95)	7.41 (9.40)	7.49 (8.68)

*Note.* PTSD = posttraumatic stress disorder; BDI = Beck Depression Inventory; *M* = mean; *SD* = standard deviation; IES-R = Impact of Event Scale-Revised; CAPS = Clinician-Administered PTSD Scale.

symptom clusters. The fairly high values of standard deviations in relation to the values for the means for all measures are consistent with what one would expect from a community sample exposed to a traumatic event wherein response to the trauma varies greatly and a considerable minority of individuals have more severe reactions to the event. On average, symptoms of both depression and PTSD declined over time. Values of the standard deviations suggest that the dispersion of scores remained relatively stable, with a slight tendency for a decrease in the fourth time class. Together with the reduced values of the means, this might imply that participants, as a group, generally tended toward wellness or recovery over time, albeit with a core of victims sustaining high distress.

### Bivariate Latent Difference Score Models

Tables 2–5 present results for the four bivariate latent difference score models examining associations between depression and PTSD intrusion, depression and PTSD avoidance, depression and PTSD numbing, and depression and PTSD

TABLE 2  
Bivariate Latent Difference Score Model for Depression and PTSD Intrusion

<i>Fit indices</i>		
$\chi^2/df$		98.08/46
CFI		.94
TLI		.94
RMSEA		.08 (90% CI = .06–.10)
<hr/>		
<i>Parameter estimates</i>	<i>Depression</i>	<i>PTSD intrusion</i>
Initial status means ( $\mu_{d0}$ ; $\mu_{p0}$ )	10.20 ( <b>15.29</b> )	10.57 ( <b>14.23</b> )
Initial status variances ( $\Phi_{d0}^2$ ; $\Phi_{p0}^2$ )	64.34 ( <b>8.06</b> )	55.68 ( <b>6.12</b> )
Constant change means ( $\mu_{Sd}$ ; $\mu_{Sp}$ )	1.51 (1.79)	−5.62 (−1.70)
Constant change variances ( $\Phi_{Sd}^2$ ; $\Phi_{Sp}^2$ )	7.65 ( <b>2.19</b> )	168.14 (1.90)
Initial status with constant change ( $\Phi_{d0Sd}$ ; $\Phi_{p0Sp}$ )	12.37 ( <b>2.25</b> )	−7.37 (−0.45)
<i>Proportional change</i>		
Depression ( $\alpha_d$ )	0.12 (1.27)	
PTSD intrusion ( $\alpha_p$ )		−2.50 ( <b>−6.63</b> )
<i>Depression-PTSD intrusion associations</i>		
Initial status <i>d</i> with initial status <i>p</i> ( $\Phi_{d0p0}$ )	47.56 ( <b>6.60</b> )	
Initial status <i>d</i> with constant change <i>p</i> ( $\Phi_{d0Sp}$ )	−67.48 ( <b>−2.30</b> )	
Constant change <i>d</i> with initial status <i>p</i> ( $\Phi_{Sdp0}$ )	12.77 ( <b>2.69</b> )	
$p_{t-1} \rightarrow \Delta d_t (\gamma_d)$	−0.52 ( <b>−7.33</b> )	
$d_{t-1} \rightarrow \Delta p_t (\gamma_p)$	2.79 ( <b>4.14</b> )	
<i>Measurement model</i>		
Loading of Impact of Event Scale-Revised	1.33 ( <b>18.78</b> )	
Intercept for Impact of Event Scale-Revised	2.96 ( <b>5.17</b> )	
Error Beck Depression Inventory	13.79 ( <b>9.74</b> )	
Error Impact of Event Scale-Revised	15.68 ( <b>6.10</b> )	
Error Clinician-Administered PTSD Scale	17.66 ( <b>8.47</b> )	

*Note.* Entries in the table's lower portion are parameter estimates with associated critical ratios (CRs) in parentheses. Symbols correspond with those highlighted in the text and used in Figure 2, where *d* represents depression and *p* represents the intrusion feature of PTSD. Salient CRs greater than 1.96 appear in bold. PTSD = posttraumatic stress disorder;  $\chi^2$  = chi square; *df* = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; CI = confidence interval;  $p_{t-1} \rightarrow \Delta d_t$  = latent PTSD intrusion score predicting subsequent depression latent difference score, with equality constraints on the three coefficients;  $d_{t-1} \rightarrow \Delta p_t$  = latent depression score predicting subsequent PTSD intrusion latent difference score, with equality constraints on the three coefficients.

hyperarousal, respectively. The first four rows of each table contain information on overall model-data fit. The findings suggest adequate to reasonably good fit. The weakest model was for depression and PTSD numbing.

The lower portions of Tables 2–5 contain parameter estimates for the models and their critical ratios (CRs), where an absolute value exceeding 1.96 generally is considered indicative of a salient estimate. The initial double-column rows of parameter estimates contain findings that describe the distinct within-variable portions of the larger model separately for depression and the relevant PTSD

TABLE 3  
Bivariate Latent Difference Score Model for Depression and PTSD Avoidance

<i>Fit indices</i>		
$\chi^2/df$	82.91/46	
CFI	.94	
TLI	.94	
RMSEA	.07 (90% CI = .04–.09)	
<hr/>		
<i>Parameter estimates</i>	<i>Depression</i>	<i>PTSD avoidance</i>
Initial status means ( $\mu_{d0}; \mu_{p0}$ )	10.22 ( <b>14.58</b> )	2.07 ( <b>7.26</b> )
Initial status variances ( $\Phi_{d0}^2; \Phi_{p0}^2$ )	70.35 ( <b>7.54</b> )	5.45 ( <b>4.50</b> )
Constant change means ( $\mu_{sd}; \mu_{sp}$ )	4.05 ( <b>3.45</b> )	0.10 (0.28)
Constant change variances ( $\Phi_{sd}^2; \Phi_{sp}^2$ )	30.99 ( <b>3.00</b> )	2.48 ( <b>2.42</b> )
Initial status with constant change ( $\Phi_{d0sd}; \Phi_{p0sp}$ )	38.00 ( <b>3.97</b> )	2.37 ( <b>2.74</b> )
<i>Proportional change</i>		
Depression ( $\alpha_d$ )	−0.70 ( <b>−5.87</b> )	
PTSD avoidance ( $\alpha_p$ )	−0.85 ( <b>−5.34</b> )	
<i>Depression-PTSD avoidance associations</i>		
Initial status <i>d</i> with initial status <i>p</i> ( $\Phi_{d0p0}$ )	12.33 ( <b>5.20</b> )	
Initial status <i>d</i> with constant change <i>p</i> ( $\Phi_{d0sp}$ )	2.04 (0.92)	
Constant change <i>d</i> with initial status <i>p</i> ( $\Phi_{sdp0}$ )	5.32 ( <b>2.00</b> )	
$p_{t-1} \rightarrow \Delta d_t(\gamma_d)$	0.07 (0.15)	
$d_{t-1} \rightarrow \Delta p_t(\gamma_p)$	0.16 ( <b>4.49</b> )	
<i>Measurement model</i>		
Loading of Impact of Event Scale-Revised	2.40 ( <b>11.50</b> )	
Intercept for Impact of Event Scale-Revised	2.83 ( <b>4.92</b> )	
Error Beck Depression Inventory	13.05 ( <b>7.47</b> )	
Error Impact of Event Scale-Revised	9.12 ( <b>7.05</b> )	
Error Clinician-Administered PTSD Scale	8.96 ( <b>10.67</b> )	

*Note.* Entries in the table's lower portion are parameter estimates with associated critical ratios (CRs) in parentheses. Symbols correspond with those highlighted in the text and used in Figure 2, where *d* represents depression and *p* represents the avoidance feature of PTSD. Salient CRs greater than 1.96 appear in bold. PTSD = posttraumatic stress disorder;  $\chi^2$  = chi square; *df* = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; CI = confidence interval;  $p_{t-1} \rightarrow \Delta d_t$  = latent PTSD avoidance score predicting subsequent depression latent difference score, with equality constraints on the three coefficients;  $d_{t-1} \rightarrow \Delta p_t$  = latent depression score predicting subsequent PTSD avoidance latent difference score, with equality constraints on the three coefficients.

symptom cluster. Across all models, the estimates of the initial status means for depression and PTSD symptom clusters were, as expected, different from 0, and the dispersion or variance estimates of initial status were likewise consistently significant (note bolded CR values > 1.96 across all four tables). The constant change mean for depression with PTSD avoidance (Table 3, CR = 3.45) and with PTSD hyperarousal (Table 5, CR = 4.00), and the constant change variance for depression with intrusion (Table 2, CR = 2.19), with avoidance (Table 3, CR = 3.00), and with hyperarousal (Table 5, CR = 2.52) were noteworthy in

TABLE 4  
Bivariate Latent Difference Score Model for Depression and PTSD Numbing

<i>Fit indices</i>		
$\chi^2/df$	101.89/43	
CFI	.91	
TLI	.90	
RMSEA	.09 (90% CI = .07–.11)	
<hr/>		
<i>Parameter estimates</i>	<i>Depression</i>	<i>PTSD numbing</i>
Initial status means ( $\mu_{d0}; \mu_{p0}$ )	10.23 ( <b>14.27</b> )	8.05 ( <b>5.89</b> )
Initial status variances ( $\Phi_{d0}^2; \Phi_{p0}^2$ )	74.81 ( <b>7.45</b> )	47.49 ( <b>2.92</b> )
Constant change means ( $\mu_{Sd}; \mu_{Sp}$ )	0.05 (0.02)	—
Constant change variances ( $\Phi_{Sd}^2; \Phi_{Sp}^2$ )	0.43 (0.14)	—
Initial status with constant change ( $\Phi_{d0Sd}; \Phi_{p0Sp}$ )	−1.51 (−0.68)	—
<i>Proportional change</i>		
Depression ( $\alpha_d$ )	−7.71 (−0.52)	
PTSD numbing ( $\alpha_p$ )		5.38 (0.37)
<i>Depression-PTSD numbing associations</i>		
Initial status <i>d</i> with initial status <i>p</i> ( $\Phi_{d0p0}$ )	59.47 ( <b>4.87</b> )	
Initial status <i>d</i> with constant change <i>p</i> ( $\Phi_{d0Sp}$ )	—	
Constant change <i>d</i> with initial status <i>p</i> ( $\Phi_{Sdp0}$ )	−1.28 (−0.69)	
$p_0 \rightarrow \Delta d_1(\gamma_{d1})$	9.42 (0.50)	
$p_1 \rightarrow \Delta d_2(\gamma_{d2})$	10.88 (0.53)	
$p_2 \rightarrow \Delta d_3(\gamma_{d3})$	10.68 (0.52)	
$d_0 \rightarrow \Delta p_1(\gamma_{p1})$	−4.52 (−0.39)	
$d_1 \rightarrow \Delta p_2(\gamma_{p2})$	−3.82 (−0.37)	
$d_2 \rightarrow \Delta p_3(\gamma_{p3})$	−3.87 (−0.37)	
<i>Measurement model</i>		
Loading of Impact of Event Scale-Revised	0.22 ( <b>5.69</b> )	
Intercept for Impact of Event Scale-Revised	2.18 ( <b>9.20</b> )	
Error/Specific Variance Beck Depression Inventory	10.72 ( <b>3.44</b> )/4.36 (1.13)	
Error/Specific Variance Impact of Event Scale-Revised	8.55 ( <b>13.74</b> )/2.42 ( <b>4.83</b> )	
Error/Specific Variance Clinician-Administered PTSD Scale	24.86 ( <b>8.77</b> )/10.92 ( <b>3.12</b> )	

*Note.* Entries in the table's lower portion are parameter estimates with associated critical ratios (CRs) in parentheses. Symbols correspond with those highlighted in the text and used in Figure 2, where *d* represents depression and *p* represents the numbing feature of PTSD. Salient CRs greater than 1.96 appear in bold. PTSD = posttraumatic stress disorder;  $\chi^2$  = chi square; *df* = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; CI = confidence interval;  $p_0 \rightarrow \Delta d_1$  = first latent PTSD numbing score predicting subsequent depression latent difference score;  $p_1 \rightarrow \Delta d_2$  = second latent PTSD numbing score predicting subsequent depression latent difference score;  $p_2 \rightarrow \Delta d_3$  = third latent PTSD numbing score predicting subsequent depression latent difference score;  $d_0 \rightarrow \Delta p_1$  = first latent depression score predicting subsequent PTSD numbing latent difference score;  $d_1 \rightarrow \Delta p_2$  = second latent depression score predicting subsequent PTSD numbing latent difference score;  $d_2 \rightarrow \Delta p_3$  = third latent depression score predicting subsequent PTSD numbing latent difference score.

TABLE 5  
Bivariate Latent Difference Score Model for Depression and PTSD Hyperarousal

<i>Fit indices</i>		
$\chi^2/df$	83.68/46	
CFI	.96	
TLI	.96	
RMSEA	.07 (90% CI = .04–.09)	
<hr/>		
<i>Parameter estimates</i>	<i>Depression</i>	<i>PTSD hyperarousal</i>
Initial status means ( $\mu_{d0}; \mu_{p0}$ )	9.76 <b>(14.68)</b>	10.53 <b>(13.62)</b>
Initial status variances ( $\Phi_{d0}^2; \Phi_{p0}^2$ )	65.57 <b>(7.88)</b>	67.93 <b>(6.33)</b>
Constant change means ( $\mu_{sd}; \mu_{sp}$ )	4.15 <b>(4.00)</b>	0.46 (0.38)
Constant change variances ( $\Phi_{sd}^2; \Phi_{sp}^2$ )	23.05 <b>(2.52)</b>	35.40 <b>(2.05)</b>
Initial status with constant change ( $\Phi_{d0sd}; \Phi_{p0sp}$ )	32.89 <b>(3.73)</b>	12.77 <b>(1.98)</b>
<i>Proportional change</i>		
Depression ( $\alpha_d$ )	−0.21 (−0.90)	
PTSD hyperarousal ( $\alpha_p$ )	−1.66 <b>(−5.40)</b>	
<i>Depression-PTSD hyperarousal associations</i>		
Initial status <i>d</i> with initial status <i>p</i> ( $\Phi_{d0p0}$ )	54.94 <b>(6.87)</b>	
Initial status <i>d</i> with constant change <i>p</i> ( $\Phi_{d0sp}$ )	−12.57 (−1.35)	
Constant change <i>d</i> with initial status <i>p</i> ( $\Phi_{sdp0}$ )	29.67 <b>(4.23)</b>	
$p_{t-1} \rightarrow \Delta d_t (\gamma_p)$	−0.41 <b>(−2.34)</b>	
$d_{t-1} \rightarrow \Delta p_t (\gamma_d)$	1.56 <b>(4.05)</b>	
<i>Measurement model</i>		
Loading of Impact of Event Scale-Revised	0.96 <b>(20.19)</b>	
Intercept for Impact of Event Scale-Revised	1.57 <b>(3.34)</b>	
Error Beck Depression Inventory	13.75 <b>(9.25)</b>	
Error Impact of Event Scale-Revised	7.97 <b>(6.05)</b>	
Error Clinician-Administered PTSD Scale	23.32 <b>(9.38)</b>	

*Note.* Entries in the table's lower portion are parameter estimates with associated critical ratios (CRs) in parentheses. Symbols correspond with those highlighted in the text and used in Figure 2, where *d* represents depression and *p* represents the hyperarousal feature of PTSD. Salient CRs greater than 1.96 appear in bold. PTSD = posttraumatic stress disorder;  $\chi^2$  = chi square; *df* = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; CI = confidence interval;  $p_{t-1} \rightarrow \Delta d_t$  = latent PTSD hyperarousal score predicting subsequent depression latent difference score, with equality constraints on the three coefficients;  $d_{t-1} \rightarrow \Delta p_t$  = latent depression score predicting subsequent PTSD hyperarousal latent difference score, with equality constraints on the three coefficients.

the prediction of depression latent difference scores. The positive values for depression's constant change component suggest that, even in the presence of prior depression and the relevant feature of PTSD, unknown and unmeasured factors still work to exacerbate depression symptom severity. This tendency is offset somewhat in the case of depression with PTSD avoidance (Table 3), as indicated by the strong and negative effect of prior status, reflected in depression's proportional change coefficient ( $-0.70$ ,  $CR = -5.87$ ). For this model,

elevated BDI scores are expected to be followed by a decrement in BDI scores at the next occasion. In contrast, the constant change component for PTSD, on average, played no appreciable role in the models involving intrusion, avoidance, and hyperarousal, though there is some evidence for individual differences in this parameter estimate for avoidance and hyperarousal. Prior status, as represented by the strong and negative proportional change coefficients for intrusion (Table 2,  $-2.50$ ,  $CR = -6.63$ ), avoidance (Table 3,  $-0.85$ ,  $CR = -5.34$ ), and hyperarousal (Table 5,  $-1.66$ ,  $CR = -5.40$ ), therefore appears to be the important predictor of subsequent change in PTSD symptoms. Those with higher scores on measures of any of these features of PTSD would be expected to score lower on those measures at subsequent assessment. Note that no values related to constant change for PTSD are reported for the depression-PTSD numbing model in Table 4. Here, to achieve convergence, the constant change component for numbing was deleted altogether, and the value of the proportional change coefficient was negligible.

Also appearing in the lower portions of Tables 2–5 are parameter estimates that directly address the linkage or comorbidity between depression and each PTSD symptom cluster. According to the guiding bivariate model (again, see Figure 2), these associations partially are manifest in the covariance between initial status for depression and initial status for the focal PTSD symptom cluster, in the covariance between initial status for depression and the constant change component for the PTSD symptom cluster, and in the covariance between the constant change component for depression and initial status for the PTSD symptom cluster. It is important, and most germane to this study, that covariation between depression and PTSD also is represented by the cross-variable lagged effects or coupling coefficients, that is, in the regressions of the latent difference scores for one variable on the antecedent latent scores for the other variable. Specifically, change in depression was regressed on prior status on each PTSD symptom cluster (e.g., the  $\gamma_{d1}$  coefficient quantifying the strength of  $p_0 \rightarrow \Delta d_1$ ), controlling for autoregressive effects and nonstationarity in depression. Analogously, change in each PTSD symptom cluster was regressed on prior status on depression (e.g., the  $\gamma_{p1}$  coefficient quantifying the strength of  $d_0 \rightarrow \Delta p_1$ ), controlling for autoregressive effects and nonstationarity in that PTSD symptom cluster. For clarity, we recapitulate the equations developed previously:

$$\Delta d_t = \alpha_d d_{t-1} + \beta_d S_d + \gamma_{dt} p_{t-1} \text{ and } \Delta p_t = \alpha_p p_{t-1} + \beta_p S_p + \gamma_{pt} d_{t-1}.$$

For each of the four models, there were six such regressions, three for the regression of change in depression on its internal sources and prior PTSD and three for the regression of change in PTSD on its internal sources and prior depression. For the models involving depression with intrusion, avoidance, and hyperarousal, equality constraints on these cross-variable coupling coefficients yielded more parsimonious and generally better fitting models.

To first address the findings related to the prediction of change in depression ( $\Delta d_t$ ), examination of Tables 2–5 indicates that PTSD symptom severity is a strong and negative predictor of subsequent change in depression over all three time intervals for the models involving depression and intrusion (Table 2,  $CR = -7.33$ ) and depression and hyperarousal (Table 5,  $CR = -2.34$ ). Thus, regarding intrusion and hyperarousal symptoms, individuals with higher scores on PTSD at the earlier timepoint are expected to have less positive change or a decrease in depression symptom severity over the subsequent time interval. In contrast, for the models involving avoidance (Table 3) and numbing (Table 4), there is no apparent unique association between prior PTSD symptom severity and subsequent change in depression symptom severity.

Turning to the prediction of change in PTSD symptom severity ( $\Delta p_t$ ), there is a fairly high degree of consistency in findings for the models involving depression with PTSD intrusion (Table 2), avoidance (Table 3), and hyperarousal (Table 5). All three depression-to-change in PTSD partial regression coefficients exceeded the 1.96 criterion ( $CRs = 4.14, 4.49, \text{ and } 4.05$ ). Thus, concerning the prediction of change in intrusion, avoidance, and hyperarousal symptom severity, individuals with higher scores on depression are expected to exhibit less negative change or an increase in PTSD symptom severity over the following time interval. As with associations between prior numbing and change in depression, the converse associations between prior depression and change in numbing were trivial and nonsignificant across all time intervals.

Finally, each of the four tables of results presents information regarding the measurement model. Because there were two indicators of PTSD for each symptom cluster and given the constraints for metric invariance, single values for IES-R loading and intercept and their  $CRs$  are provided. For the model related to PTSD numbing, estimates of specific variance and their  $CRs$  also are given. In all cases, the  $CRs$  exceed the 1.96 standard.<sup>3,4</sup>

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<sup>3</sup>In addition to the four-factor representation of the structure of PTSD used in this article, an alternative four-factor structure was proposed by Simms et al. (2002). In this alternative model, the intrusion and avoidance symptom clusters remain the same, but a subset of items from the hyperarousal cluster are shifted to the numbing cluster to form what Simms et al. called *dysphoria* and a truncated hyperarousal cluster. When bivariate latent difference score analyses were performed to examine associations between depression and these modified symptom clusters, the findings supported positive and significant depression to change in dysphoria cross-variable links; dysphoria to change in depression links were nonsignificant, as were the cross-variable associations between depression and the reduced hyperarousal cluster. Results of these supplemental analyses are available from Daniel W. King.

<sup>4</sup>We also considered the possibility of a model incorporating nonlinear change. In particular, we consulted Hamagami and McArdle's (2006) model of differences between differences intended to accommodate shifts in slopes to capture acceleration or nonlinear/quadratic trajectories in the data. We evaluated this latent acceleration approach for the depression and intrusion, depression and avoidance, and depression and hyperarousal associations. In each case, the latent acceleration model did not fit as well as the latent difference score model, and thus the latter model was preferred.

## DISCUSSION

The aim of this article was to catalog temporal dependencies between posttrauma depression and PTSD symptom severity using a method that allows for explicit examination of directional influences of mutual sequential difference scores. With a sample of adult emergency room admittees who experienced a traumatic event, we explored changes in depression (measured by the BDI) and PTSD (indexed by the IES-R and CAPS) from the period immediately following exposure to the trauma through the stage of potential chronicity. Dynamic change analysis focused on three time intervals, approximately 9–36 days following exposure, 37–131 days following exposure, and 132–192 days following exposure (using the medians for time classes; see Table 1), which formed the basis for the three latent difference scores on each variable. We thus documented the extent to which depression symptom severity might influence change in PTSD symptom severity and the extent to which PTSD symptom severity might influence change in depression symptom severity. In summary, results showed depression-to-change in PTSD links to be positive and strong, suggesting that level of depression is antecedent to positive shifts or worsening in PTSD. Considering the reverse direction of influence, negative and strong PTSD-to-change in depression associations obtained for the symptom clusters of intrusion and hyperarousal. For the model involving avoidance, no PTSD-to-change in depression associations were found. Finally, for the depression and numbing model, all cross-variable lagged associations were negligible, essentially indicating no unique cross-variable predictors of change in either depression or numbing.

With regard to understanding trends toward wellness or chronicity in PTSD following trauma exposure, the results for the models incorporating depression with PTSD intrusion, avoidance, and hyperarousal are consistent. Again, we reference the three influences in the general equation for predicting change in PTSD: (a) autoregressive effects or proportional change, (b) nonstationarity or constant change, and (c) cross-variable lagged effects. For each model, the negative and salient value for the PTSD proportional change coefficient demonstrates the expected tendency for PTSD symptoms to abate over time (e.g., L. A. King et al., 2003; Koss & Figueredo, 2004); possible mechanisms for this effect may be habituation to traumatic memories or extinction of a conditioned emotional fear response upon repeated exposure to event reminders. The constant change coefficient signals the effect of potential unknown or uncontrolled factors that impact change, such as individual differences in resilience: active coping skills, support and information seeking, and cognitive flexibility for perspective-taking and meaning-making. In this case, the values of the constant change coefficient show it to be less essential for all PTSD components. But the positive and important impact of prior depression on subsequent worsening of PTSD offsets the anticipated diminution of symptoms. In other words, higher levels

of depression work to exacerbate PTSD intrusion, avoidance, and hyperarousal symptoms, even in the presence of a general trend toward wellness.

To use just one example for demonstration, we return to the equation for the latent difference score for PTSD and Table 2 parameter estimates for the depression-PTSD intrusion model, where the proportional change coefficient is a significant  $-2.50$ , the constant change mean is a nonsignificant  $-5.62$ , and the coefficient for the influence of prior depression on subsequent change in PTSD intrusion is a significant  $2.79$ :

$$\Delta p_t = \alpha_p p_{t-1} + \beta_p S_p + \gamma_p d_{t-1}$$

$$\Delta p_t = -2.50 p_{t-1} - 5.62 S_p + 2.79 d_{t-1}.$$

Although autoregression or proportional change and nonstationarity or constant change are represented in terms of to-be-expected decreasing PTSD symptoms across time (both with negative signs, albeit only the proportional change coefficient achieving significance), prior depression intimates a worsening of subsequent PTSD symptom severity (positive sign), even in the presence of these other effects. Thus, the equation would suggest that “high enough” depression scores ( $d_{t-1}$ ) can offset the expected trend toward wellness (due to the aforementioned habituation, extinction, or intrapersonal resilience factors) and lead to gains in PTSD intrusion scores ( $\Delta p_t$ ).

To further offer insight into the interpretation of the metrics in predicting  $\Delta p_t$ , we reference well-known BDI guidelines for depression intensity and project the degree of change in subsequent PTSD symptom severity indexed by the CAPS. With the aforementioned equation and data from the second assessment of depression and PTSD, we predicted PTSD intrusion scores and then change in PTSD intrusion scores using three representative levels of depression intensity. With mild depression ( $BDI = 5$ ), CAPS intrusion scores decreased  $-4.86$  points; with moderate depression ( $BDI = 20$ ), an increase of only  $0.24$  in CAPS intrusion scores was predicted; and for severe depression ( $BDI = 35$ ), a  $5.34$  jump in CAPS intrusion scores was expected. Given that the latent intrusion variable had a standard deviation of  $9.15$  at the second assessment, it appears that mild depression would yield a decrease in intrusion symptoms by about one half of a standard deviation unit, whereas severe depression would portend an increase of almost six tenths of a standard deviation unit, thus overwhelming other factors that might reduce symptom severity.

Why might level of depression portend an intensification in PTSD symptoms? Perhaps posttrauma depressive ruminations are reformulated by trauma victims as powerful reminders of the event and other reexperiencing phenomena. If intense and unrelenting, depressive ruminations concerning the traumatic event might lead directly to increases in intrusion symptoms, the hallmark feature of PTSD, as well as attempts to avoid reminders of the event. This is one

mechanism that might explain the results of this study, and it is clearly applicable to the consistent finding of depression-to-change in intrusion and depression-to-change in avoidance associations. As regards hyperarousal, perhaps a similar mechanism operates wherein the sleep and concentration difficulties and possible restlessness that characterize depressed persons who have experienced a trauma then trigger the more severe and classic hyperarousal feature of PTSD, especially tendencies toward a startle response and extreme vigilance. A more general possible mechanism is that high levels of depression may signify dysphoria, a sense of helplessness, and loss of mastery, which would attenuate an individual's motivation for extinction of PTSD symptoms via active support-seeking, self-disclosure, and other self-efficacious behaviors that ameliorate stress responses. An initial dysphoric/depressive response to trauma may be subsequently articulated into the classic symptoms of PTSD.

In the models for depression with avoidance and hyperarousal, the significant constant change coefficients for the prediction of change in depression suggest factors unspecified in the model might be contributing to increases in depression symptoms (note positive signs). Such influences might include previous adverse experiences, additional life stressors, posttrauma survivor guilt and sense of failure, or loss of personal resources. All other salient coefficients in the prediction of change in depression, that is, proportional change for the model involving avoidance and cross-variable lagged effects for the models involving intrusion and hyperarousal, point to an expected diminution in depression (note negative signs). Unlike for the prediction of change in PTSD, where prior depression heralds an increase in PTSD symptom severity, the cross-variable dynamic in PTSD's prediction of shifts in depression follows the trend toward recovery. Hence, although depression is arguably a risk factor for, or unique contributor to, increases in PTSD, PTSD does not appear to be a risk factor for, or unique contributor to, increases in depression. The mechanisms that underlie this pattern may be grounded in the extent to which the two conditions are differentially laden with predispositional versus environmental influences within a diathesis-stress model. Although both confer a mix of genetic- and stressor-based influences, depression, if considered representative of a predisposing temperament, is etiologically antecedent to PTSD, whereas PTSD, a consequence of exposure to an environmental event (and other risk factors), is not etiologically antecedent to depression.

The model involving depression and PTSD numbing did not offer any clear evidence suggesting a dynamic interplay between the two variables other than the rather simplistic observation of a significant association between the two variables at initial status. There are two possibilities in explaining this relative dearth of findings. The first is the overlap or similarity in the constructs of depression and numbing; probably more so than any other feature of PTSD, numbing—lack of affect, withdrawal from relations and social activities, sense

of no future—seems to carry a good degree of commonality in meaning with depression. Thus, unique contributions to change in one or the other would be difficult to demonstrate. Second, a close examination of the item content of the IES-R might suggest a distinct numbing factor was not well represented. Of the three items identified for this factor, only one could convincingly be called a direct indicator of numbing (“feelings . . . were . . . numb”).

This study is similar to the work of D. J. Erickson et al. (2001) in that both examined the interplay between depression and PTSD symptom severity using panel designs. Whereas the D. J. Erickson et al. study of Gulf War I veterans employed multiple regression and endorsed bidirectional associations between depression and PTSD, the current study of emergency room patients generally supported a more unidirectional depression-to-increases in PTSD process. It is possible that the difference in findings simply lies with the aforementioned contrast of statistical approaches. In the multiple regression analyses, only prior status on depression is partialled before the effect of PTSD on depression is considered, whereas the dual change latent difference score analyses represent change in depression as a function of both prior status on depression and unexplained change in depression, both partialled before the effect of antecedent PTSD is examined. Thus, in the latent difference score model, the effect of PTSD on change in depression is net of PTSD’s contribution to unexplained change in depression. It may be, therefore, that the PTSD-to-depression cross-lagged coefficients reported by D. J. Erickson et al. would become less pronounced in the presence of another predictor, unexplained change in depression, and that the dominant influence would be from prior depression to PTSD.

Moreover, the timing of assessments vis-à-vis trauma exposure in the two studies is important to recognize. For D. J. Erickson et al. (2001), initial assessment was upon arrival back to the United States from the war zone, roughly 6 months after the close of hostilities and putatively 6 months after exposure to severe war-related stressors. For the current study, the initial assessment averaged 9–10 days following the event, and the average for the last time class was in the vicinity of 200 days or 6 months postexposure. It is possible, therefore, that emergent PTSD demonstrates a pattern of relationships with depression different from a more chronic form of PTSD, hence accounting for a different dynamic process.

Unlike prior studies that examined the temporal sequencing of depression and PTSD from an epidemiologic, lifetime, condition-based perspective (e.g., Breslau et al., 2000; Kessler et al., 1995), this study observed the dynamics of depression and PTSD cross-variable symptom relationships within individuals in the 1st year following a trauma. Our data do not address whether a preexisting diagnosis of one condition makes one more vulnerable to the onset of the other condition. Then again, given that depression and PTSD are highly likely to co-occur subsequent to trauma, the findings address process and attempt to uncover mechanisms that explain observed comorbidity and govern exacerbation

or diminution of posttrauma sequelae. In turn, they provide an endorsement for the broader conceptualization of response to traumatic events called for by Shalev (2002, 2003). In the present study, the structure underlying the observed covariation or comorbidity suggests that levels of depression anticipate subsequent change in PTSD symptom severity. Those for whom depression symptoms are high are more likely to exhibit an increase in consequent PTSD symptoms, and those low in depression would be expected to experience less of a subsequent increase or a decrease. Therefore, monitoring levels of depression symptom severity can alert to the possibility of ensuing spikes in PTSD symptom severity with the obvious implications for intervention and treatment.

A final note on the method for this study: The latent difference score model was developed for equal intervals of time between assessments. Equal time intervals permits the generalization of conclusions from the model to intervals at any location on the time dimension. This equality can be expressed not only in actual time but also in latent or psychological time, allowing for the metric to be transformed into logarithmic, exponential, or other functions as long as they can be justified in some psychological sense. As in most longitudinal trauma research, this study's actual times of data collection deviated from the intended data collection design calling for assessments at 1 week, 1 month, and 4 months. Participants were assigned to time classes to minimize dispersion of the times of assessment within classes and maximize covariance coverage or the amount of available information that could be used. Unfortunately, the time structure of the data could not accommodate any representation of equal time intervals. Using individual power-polynomials, we reconstructed the data for each participant in two alternative ways, one with assessments at 1 week, 1 month, 6 months, and 1 year, and then every week for 48 weeks, and fit the latent difference score models to each data set. Although the pattern of overall results were similar to those reported here, values of parameter estimates were not. We therefore conclude that the findings of this study should be considered specific to the actual time intervals used and caution against overgeneralizing to other time intervals.

On the positive side, the time intervals that were used in this study have a rough correspondence to conceptual models of the unfolding of psychological distress, specifically PTSD, following exposure to a traumatic event. That is, the progression of the average number of days (e.g., the mean or median days for assessments assigned to each of the time classes) substantively captures critical benchmarks for the evolution of a PTSD diagnosis, with the 30-day *DSM* criterion marking the transition from acute stress response to a formal diagnosis encompassed in our first time interval; the official transition point for chronic status, 90 days, enclosed within the second time interval; and the third time interval, stretching to approximately 6 months postexposure, allowing the expression of longer term symptomatology. According to theory, then, important trends are expected to occur within the first 90 days after a traumatic event, and increased

assessments over that interval might be more sensitive to the detection of critical change and influences on that change. Additionally, please note the aforementioned comparisons with the D. J. Erickson et al. (2001) findings concerning the timing of assessments as a possible explanation for variations in patterns of associations. Future panel research of this type should strive for equal, more individually consistent, and perhaps different time intervals to permit stronger conclusions and thus enhanced generalizability over a full time spectrum.

In closing, it should be noted that no data were available regarding participants' receipt of treatment for either depression or PTSD during the course of the study. Furthermore, the sample in this study was rather heterogeneous with regard to trauma exposure. A recommendation for future research, therefore, might be to apply the latent difference score approach to understanding mechanisms related to posttrauma depression and PTSD symptom severity using other trauma populations, possibly individuals exposed to a more specifically delineated common traumatic stressor. We also suggest that follow-up research use similar design and statistical strategies with other indicators of both depression and PTSD to determine if directional associations are upheld. It would be useful to include multiple indicators of depression in future models. What's more, further intense examination of the mechanisms underlying the mutual influences of depression and PTSD could be conducted at the level of factors or symptom clusters for both. Perhaps there are subfeatures of depression that can be incorporated into similar dynamic models in conjunction with PTSD symptom clusters.

## ACKNOWLEDGMENTS

This study was supported by National Institute of Mental Health Grants R01MH68626 (Daniel W. King, principal investigator) and R01MH50379 (Arieh Y. Shalev, principal investigator). We thank our colleagues, Brett Litz, Mark Miller, and Jeffrey Knight, for their contributions to the interpretation of findings.

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